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BURNS OF THE ESOPHAGUS CAUSED BY CORROSIVE CHEMICALS

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Honored Worker of Science

Corrosive chemical burns of the esophagus belong to that class of diseases which are still treated according to outmoded methods. Thus, even today, medical men still do not practice bougienage of the alimentary tract until "complete healing," i.e., until the formation of cicatrical stenosis. Bougienage, however, becomes difficult with the formation of scar tissue and this can be avoided if patients submit themselves to bougienage prior to the setting in of stenosis of the esophagus.

More than 30 percent of the cases of corrosive chemical burns are fatal soon after the occurrence of the burn. Complete recovery occurs in less than 30 percent of the cases, and cicatrical stenoses are eventually observed in the remaining cases.

The author notes that those reporting to his clinic (Moscow Oblast Scientific Research Clinical Institute, MONIKI) were local inhabitants. In a 2-year period, there was a total of 80 cases with stenosis of the esophagus, 46 women, 25 men, and nine children. All the cases showed evidence of advanced stenosis of the esophagus caused by caustic alkali burns. In spite of explicit directions given to otolaryngologists to the effect that patients must be sent to the clinic while the burns are still fresh, very few cases with fresh burns arrive. In 1945, for example, only ten cases showed fresh burns (a similar situation was noticed at other institutions). During the period 1930 to 1934, a total of 122 patients with corrosive chemical burns of the esophagus were examined at the Surgical Clinic (chief of clinic, Prof V. S. Levit). Only 15 showed fresh burns while the remainder had cicatricial stenosis. It is considered that a large number of the cases with constricted alimentary tracts could have been prevented had the patients submitted to bougienage immediately after the occurrence of the burn.

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The degree of damage to the esophagus depends on several factors other than resistance barriers and reactive mechanisms of the organism, for example: (1) the nature of the substance which had been swallowed (sulfuric acid and caustic soda cause deeper damage than either acetic acid or aqua ammonia.), (2) the amount of corrosive substance swallowed, (3) concentration of the solution and the duration of its action, and (4) consistency of the solution (more viscous liquids pass through the alimentary tract slowly and consequently cause severer burns).

Generally there are four stages in burns of the esophagus: (1) hyperemia and edema, (2) formation of ulcers, (3) granulation, and (4) cicatrication.

Hyperemia and edema of the mucous membrane occur during the initial stage. The edema is the result of absorption of the corrosive substance by the cells and the inflammatory reaction. In minor cases the epithelium becomes loose, then peels off. Surface erosion heals by poliferation of the epithelium from the edges, and generally there is no scar tissue. Nevertheless, slight irritation of the mucous membrane of the esophagus occurs even if the patient has expectorated the corrosive which was taken accidently. It is noticed that after expectoration there occurs an "empty" swallow, by which invariably sputum mixed with the corrosive agent enters the esophagus.

In cases of severe corrosion, the epithelium might be detached in the form of a film or even in the form of a tube. In exceptionally severe burns, particles of the mucous membrane or even muscular tissue are detected in the expectorated matter (oesophagitis dissecans). The separation of necrotic matter generally occurs 5 to 10 days after poisoning, in some cases even later.

The first stage overlaps the second stage without any well defined boundary. After the initial scab falls off, a general suppuration sets in due to secondary infection of the ulcer. The surface of the ulcer is uneven, is covered by yellow or hemmorhagic fibrous exudate, and varies in depth. Cavities are observed and frequently suppurative processes occur in the walls of the alimentary tract.

In the third stage the surface of the ulcer is covered with granulations. This indicates the disappearance of necrotic tissues. Subsequently granular tissue is replaced by connective tissue and the epithelium is regenerated. From the clinical standpoint granulation occurs in the middle of the second week following exposure, simultaneously with cessation of dysphagia. The patient regains normal swallowing and considers himself cured. The inexperienced physician will consider the patient's condition satisfactory and will generally send him home without further examination. However, after 8 to 12 weeks the patient returns to the hospital, with stenosis. This third stage is the most dangerous, because it fools both the physician and the patient. Systematic and persistent bougienage must be begun during this period.

The mechanism of cicatrication is similar in all cases (fourth period). The ulcerous region is usually circular in shape. Granulations which cover the ulcer join together and cover the surface. Gradually granular tissue is replaced by fibrous connective tissue--either hard or soft scar tissue--depending on the depth of the ulcer. It is during this period that complete primary stoppage of the esophagus may occur, but, fortunately, such cases are rare. In the great majority of cases, stoppage of the esophagus is a secondary effect. Even in cases of severe burn there remains an eccentric passage with a diameter of 1 to 2 millimeters. Subsequently, food widens out a section so that it looks like a bag, where the food collects, ferments, and decomposes. This is the source of a new infection, leading in some cases to complete secondary stoppage. Of all the cases examined at KOMKI only two had complete secondary stoppage. In rare cases there might be two constrictions (at the time of writing of this article there was one such patient--female--in the clinic).

This patient has one stenosis in the form of a thin diaphragm in the upper third of the esophagus (the first physiological constriction), and the other stenosis is located at the diaphragmal physiological constriction of the esophagus. It must be noted, however, that in many cases where clinical examinations identify two constrictions, surgery will reveal only one, whose top and bottom are so separated that it seems like two separate stenoses.

Stenoses usually occur at the point of physiological contraction of the esophagus: at the entrance of the esophagus; in the region of bifurcation; and over the esophagus opening of the diaphragm. However, the location of the stricture may vary greatly, and can be found even between physiologic contractions.

Symptoms are well defined during the first and second stage, but are not evident during the third stage, while during the fourth stage symptoms appear in the form of a gradual or sudden stenosis.

During the initial stage it is necessary to differentiate between subjective, objective, and general symptoms. Immediately after exposure to corrosive chemicals there occur pains in the mouth, glottis, chest, and around the lips. Pains are felt sharply in the dorsal region. Swallowing becomes difficult, and often impossible. Any attempt to swallow, even liquids, intensifies the pain. Patients become frightened prior to swallowing, refuse food, and suffer pangs of hunger and thirst. Objective symptoms follow soon thereafter in the form of vomiting of bloody mucus. Vomiting and even the retching reflex are accompanied by intense pain. Frequently there are hiccups. This combination of vomiting, hunger, thirst and hiccuping makes the patient very uncomfortable, and there is a noticeable increase in saliva discharge. Urine discharge decreases, and albumin as well as formative elements can be observed in the urine. The white count indicated decrease of leucocytes accompanied by a neutrophile shift to the left (alkali poisoning) or considerable leucocytosis (acid poisoning). The erythrocytes react to the general condition and indicate anisocytosis and partial disintegration. The temperature rises and there is a general depression, pallor, irritation, and sleepiness.

All of the above-mentioned symptoms disappear after the tenth or twelfth day and the patient begins to feel much better. This corresponds to the formation of the granular layer. The patient can swallow food without difficulty or pain, and again considers himself cured. At this time the attending physician should stress the necessity for treatment, particularly for carrying out bougienage.

Premature interruption of treatment 2 to 4 weeks after exposure usually results in further complications. Instead of localized pain there is a sensation of painful obstruction, which is easily localized. The swallowing of food becomes progressively more difficult, and there is no letup in this condition. At first only large mouthfuls of food do not pass, and have to be vomited. Gradually the patient is forced to chew his food thoroughly and swallow water with the food. Eventually the patient is reduced to eating mush, and finally he is able to swallow only liquids.

In cases where the constricted region is situated eccentrically, the patient often has to bend his whole body so as to accomplish swallowing. Difficulty in swallowing food is accompanied by belching, nausea, and vomiting. In cases where the stricture is in the upper part of the alimentary tract, belching and vomiting occur immediately after eating. In the event that belching and vomiting occur some time after eating, a stenosis is indicated in the lower part of the esophagus. Belching and vomiting might occur at any time and not necessarily as a result of eating.

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Prolonged stenoses result in emaciation of the patient. Secondary spasms frequently cause a sudden stoppage of the esophagus. Even in cases of temporary stoppage the patient suffers from hypochromic anemia, the color and texture of skin changes (it becomes pale, wrinkled, and clammy), and muscles become atrophied. The patient at this point may die unless he receives immediate surgical treatment.

A diagnosis of burns of the esophagus is not difficult. Treatment must be directed primarily toward saving the life of the patient, then procedures for preventing the formation of stenosis should be applied.

A patient arriving at a hospital should get an immediate morphine injection, in order to quiet him and to strengthen cardiac activity. It is almost paradoxical that at such a time caffeine or camphor have little effect, and only morphine is satisfactory. The morphine injections should be kept up for a period of 7 to 10 days. As a result, physical suffering is decreased, and hunger and thirst are diminished.

The physician should then consider the removal or neutralization of the corrosive chemical. If the patient enters a hospital within 7 hours after exposure, a gastric lavage should be carried out. Data is available which confirm the value of immediate lavage (gastric as well as of the esophagus). The effectiveness of stomach lavage is nil after 7 hours, i.e., after the absorption of the poison. Antidotes are effective only during the first few hours subsequent to exposure.

During the next few days, subsequent to arrival at the hospital, the patient is given physiological salt solution subcutaneously (500 to 1,000 cubic centimeters daily) and gastric clyses (both drop method and nutritive ones), which is followed by a tablespoonful of high-grade olive oil every 2 hours (up to 200 cubic centimeters per day). The olive oil lubricates the walls of the esophagus, washes off any dead tissue, lessens pain, protects the walls from infection which might be caused by the passage of food, promotes regeneration of tissue, decreases thirst, and also acts as a satisfactory nutrient medium. The patient finds the olive oil quite palatable, but this is understandable in view of the fact that the taste buds are severly damaged. At the same time rinsings with mild boric acid or alkali solutions, depending on the type of poison taken, so as to effect neutralization, are recommended.

After 10 to 12 days the patient feels much better, and pain decreases. The problem now is to prevent stenosis.

Bougienage has been used for many years for this purpose. Some experts (Gersuni, 1887; Manchert, 1742) recommended bougienage immediately after exposure. However, such complications as frictional ulcers and tumors in the esophagus, high temperatures, frictional erosion of the right subclavical artery, etc., were strong arguments against early bougienage. Roux, however, obtained excellent results with early bougienage in 1913 by means of improved methods.

The bougie was introduced through the nasal passage and kept in the esophagus for 20 to 30 days. Caution is necessary however, as prolonged installation of the bougie may cause irritation of even healthy mucous membranes (esophagitis). Patients who are treated with the bougie may develop inflammation resulting from prolonged installation, or the existing inflammation may be aggravated. For that reason, Roux's method has been modified to the extent that the bougie is not left in the esophagus for a prolonged time, but only for short periods.

Today the general tendency is to practice early bougienage. However, there is considerable controversy as to the exact time for starting bougienage. Some Soviet authorities (Rassudov of the Hospital imeni Filatov) recommend bougienage after the fifth day, some (Levit) after the second day, while others (Belinov) do not begin bougienage until the tenth day. The period for keeping the bougie in the patient varies from 1 to 6 hours (Petrova) to 2 to 3 days (Danilov). Clinical results, however, seem to support those authorities recommending fairly early bougienage.

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The author recommends bougienage after the tenth day. This treatment should not be started unless the patient feels relatively well. The bougie is inserted daily for 15 to 30 minutes. The thickest bougie should be used immediately. The bougienage is kept up for 4 to 6 weeks, and its use should be preceded by esophagoscopy to determine the condition of the alimentary tract.

The patient's diet is controlled as follows: (1) the first 10 days of bougienage the patient is kept on liquids and (2) after the initial period the patient is given finely chopped meat, bread, and other solids.

Early bougienage results in a high percentage of recovery, and mortality and serious complications are almost completely eliminated. Patients who already show advanced stenoses have a high mortality rate (40 to 50 percent). Other data show a mortality rate of only 10 percent. The author states that out of 80 patients who reported to his clinic only 7 percent died. This figure, however, is inaccurate because many die after they are discharged from the hospital. Many of these cases die of gradual emaciation or tuberculosis. Although statistics do not show that the patients died of cicatricial stenosis, the fatalities in question properly belong in that category.

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